Females.	*Years.*	*Months.*
Average residence in hospital of those discharged	1	7
Longest period of residence of any case	10	6
Shortest period of any case	...	1
Average period of residence of those who remained under treatment until termination of disease	2	3
Longest period of treatment of case	11	...
Shortest period	...	1
Average period of duration of patients remaining under treatment at hospital	3	4
General average of length of duration of disease of all cases observed in hospital	2	9

Of the patients admited 3 per cent. were in the first stage of paresis.
" " " 89 " " second stage.
" " " 8 " " third stage.
Periods of remission from all active symptoms of the disease occurred in 9 per ct. of cases.
Convulsive seizure occurred during the progress of the disease in . 60 " "

TERMINATION.

Termination of paresis in death in convulsion occured in . . . 30 " "
Death as a result of exhaustion in . . . . 70 " "

ARGUMENTS IN FAVOR OF THE EXISTENCE OF A SEPARATE CENTRE FOR WRITING.*

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No subject in neurology has attracted more attention or excited more discussion than agraphia; that is, whether or not the ability to write depends upon the integrity of a separate cortical centre near to, but absolutely distinct from, that of Broca's area, through which articulate speech is effected. Two diametrically opposed views are at present earnestly maintained. The first and older view is that a focal cortical centre for writing does not exist, and that the inability to write is as much the result of a lesion of Broca's centre as is the inability to speak. A lesion of this centre, therefore, always excites motor aphasia or agraphia. This is the one advocated by the earlier observers—Trousseau,[1] Gairdner, Wernicke, Lichtheim, and Hughlings Jackson—and now strenuously supported by Dejerine, Mirallie,[2] and Wyllie, in Europe, and Collins[3] in America. The second view, on the contrary, maintains that the act of writing is independent of the faculty of expression by articulate speech, and its loss is due to the destruction of a cortical centre, distinct from the motor speech or arm centres, and its seat is probably located at the base of the second left frontal convolution. The supporters of this view believe that agraphia does occur as a distinct entity; that it may be unaccompanied by motor aphasia, and that when the two defects occur together both the motor speech and the writing centres or the association tracts connecting these centres are involved.* This view is supported by Exner, Marcé,[4] Ogle,[5] Charcot, Kussmaul, Oppenheim,[6] Bastian,[7] Beevor,[8] Bramwell,[9] and Collier, in Europe, and Eskeridge, Pershing,[10] Mills, Fränkel, and the author,[11] in this country. In support of this view, I have the following propositions to offer, with the discussion of each in their order:

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* Read at the Annual Meeting of the New York State Medical Society, January, 1903.

First, that sufficient clinicopathological data exist to show that motor aphasia and agraphia do not necessarily exist together, and that complete destruction of Broca's centre does not necessarily cause agraphia.

Second, that when motor aphasia and agraphia coexist, the lesion probably involves the bases of both the second and the third left frontal convolutions in the right-handed or the association tracts connecting these centres with the visual or auditory word areas.

Third, isolated motor agraphia, though exceedingly rare, does occur and depends upon a lesion of the base of the second left frontal gyrus in the right-handed.

In connection with the first proposition, it is very interesting to introduce Trousseau's own words in reference to the subject. He says: "The inability to write is proportionate to the inability to speak. Aphasics write as badly as they speak, and those who do not speak at all are absolutely incapable of writing." Marcé, on the contrary, in 1856, stated that "the centres of co-ordination for spoken and written words are different and separated from each other in position, since twice the patients could write freely when they were quite unable to speak. It is independent of the mobility of the hand, since when the hand has preserved all its powers writing has been found to be impossible."

In support of the first proposition and to show the fallacy of Trousseau's reasoning, I will introduce abstracts of the following cases, one of the most typical of which was Trousseau's own case.

He tells of a robust young civil functionary who, in an attack of unconsciousness, had entirely lost the power of speech. There was no paralysis of any parts of the body. He could execute all the movements of the tongue and lips with the greatest facility. He was able to discharge the duties of his office, notwithstanding the loss of speech, as he could transact all his business in writing. He delivered to Trousseau an exhaustive history of his case which had been written out by himself.[12]

Oppenheim[13] reports another interesting case of a painter, who for seventeen years following an apoplectic attack was completely aphasic and could utter nothing save the words "yes" and "no," but could give a written account of his entire illness and could paint beautifully.

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* Dr. Beevor states in a personal communication that his case is one of pure motor agraphia with slight right-sided hemiplegia unaccompanied with aphasia, word-blindness, or word-deafness.

Idelson,[14] Oppenheim's assistant, has recorded a case of a patient who had been cured by inunctions from a left-sided hemiplegia, hemianopsia, and hemianæsthesia, who after a new attack became completely aphasic and was not able to utter a single word or syllable, but was able to write perfectly during his entire illness.

Charcot,[15] records the case of a woman who was under his care at La Salpêtrière. She had been seized with aphasia, but there existed no trace of paralysis, either of movement or of sensibility. In his patient aphasia was the sole morbid symptom, and atrophy of the left third frontal convolution was the only lesion revealed by the autopsy. This was believed to be due to an old occlusion or narrowing of the external and inferior frontal branch of the middle cerebral artery.

Bramwell,[16] in the *Lancet*, May 22, 1897, describes a case in which acute softening, confined to Broca's convolution, produced complete motor aphasia with little or no agraphia. The absence of any marked agraphia, according to Bramwell, was forcibly illustrated by the fact that the patient, who was unable to answer questions in vocal speech, carried a note-book about with him for the purpose of answering questions and carrying on a conversation in writing. Bramwell explicitly states that the case seemed totally opposed to the views to which the great majority of authorities on aphasia held, that destruction of Broca's convolution necessarily produces agraphia and that, as Gairdner long ago argued, the degree of agraphia is usually proportionate to the motor aphasia. It seems to support the view advocated by Bastian, which he also, independently, arrived at, *i. e.*, that the nervous impulses which set the motor writing centre into action do not necessarily pass through the motor speech centre in order to reach the writing centre, and that destruction of the motor speech does not usually give rise to agraphia.

Prof. C. K. Mills has sent me the notes, with photographs of the brain, of a very interesting case, supporting the theory of the dissociation of agraphia from motor aphasia. The lesion, as the photographs show, was limited to Broca's convolution.

Referring to his case, he says: "The main features of this case are as follows: The patient, after an apoplectic seizure, developed a peculiar form of aphasia, apparently without any preceding paralysis or any other symptoms of focal lesion like visual blindness, hemianopsia, etc. When examined by me about nine years after the onset of the aphasia, he had almost complete inability to name persons and objects which he was able to recognize through all his special senses. He had also a marked form of paralexia or paranomia. When he attempted to read, although he understood what he was reading, he repeated an absurd formula of a few phrases. He had limited spontaneous speech, even using short sentences without concrete nouns correctly. He could write many single words correctly, sometimes misspelling, however. He held his pen or pencil correctly and wrote such words as he did write with ease and firmness. He was not a motor agraphic."

Ogle,[17] Bastian,[18] and Dickinson[19] have each reported cases in which there was right hemiplegia and aphasia, though the patients could write with their left hand. Ogle's case came to autopsy and showed a patch of softening involving the left frontal convolution and reaching downward and backward to the fissure of Sylvius. The second frontal gyrus was normal.

I will refer, briefly, to a case recently under my care, of a boy who entered the Samaritan Hospital, suffering from a depressed fracture of the frontal bone. He had several attacks of unilateral spasms of the face and complete aphasia. The depressed bone was elevated and several blood clots removed, but the aphasia remained. He nodded assent or dissent when spoken to, recognized objects, and seemed to understand, perfectly, printed or written language. There was no hemianopsia or paralysis of either arm. He was able to write his name the day following the operation and in a few days he wrote sentences, copied, and wrote correctly from dictation.

Altogether the most remarkable case on record to disprove the views of Trousseau, Dejerine, and their followers, is the one recorded by Guido Banti.[20] It will be reported here in full. A right-handed man, aged thirty-six years, who was able to read and write correctly, had a sudden apoplectic attack in 1877. Recovering consciousness in a few minutes, he was found to be suffering from right hemiplegia and loss of speech. The paralysis of the limbs disappeared the following night, although the inability to speak remained. The next day he was admitted to the hospital, and after a most careful examination, his condition was found to be as follows: The mobility of the limbs on the right side had returned to their normal condition. There was no trace of paralysis of the face or tongue. The patient made ineffectual attempts to speak, but he could not articulate a single word, not even isolated syllables. He was much affected by his mutism and sought to make himself understood by gestures. When given the necessary articles and when asked to write his name, he did it immediately. Other questions were put to him to which he replied in writing. He wrote the details and description of his illness without hesitation. Various objects were shown to him, such as pieces of money, etc., and he answered, correctly, the name of each object in writing. Then instead of giving him directions by word of mouth, I wrote them for him, in order to thoroughly convince myself that he was able to understand writing. He replied to these questions with perfect correctness. He wrote very rapidly and did not seem to hesitate for words. He made no mistakes in syntax or orthography. He could understand, equally well, ordinary writing and print, and when one spoke to him he grasped their meaning at once, and never wished to have their questions repeated. Banti wrote some simple words and asked him to repeat them aloud. This he could not do. He then pronounced some words and asked him to repeat them after him. He watched the movements of my lips and tried very hard to obey, but he never succeeded in pronouncing a single word. The patient died in February, 1882, from an aneurism of the aorta. At autopsy a patch of yellow softening was found, situated in the posterior third of the left third frontal convolution.

In summarizing the ten cases above narrated, we find that complete motor aphasia occurred in each without agraphia. In five of the cases no autopsies were had, but in each of these cases the clinical history was very plain, and there was no paralysis of the muscles of either hand. In five of the ten cases autopsies were had, namely, those of Charcot, Ogle, Banti, Bramwell, and Mills. In Charcot's case the pathological lesion was an atrophy confined to the left frontal gyrus, due to occlusion of the inferior branch of the Sylvian artery. The only symptom was complete motor aphasia. In Ogle's case the third left frontal convolution was softened, while the second left frontal convolution was found normal, and there was motor aphasia without agraphia. Banti, in his case, found as the only lesion an area of softening limited to the posterior third of Broca's convolution, involving only the cortex. This had caused in a right-handed person, perfectly able to read and write, motor aphasia without agraphia, there being no word deafness, alexia, or hemianopsia. In the very carefully reported case of Bramwell, softening limited to Broca's convolutions produced complete motor aphasia with little or no agraphia. In Mills' cases the lesion was confined to Broca's area and still there was no motor agraphia. Collins, in his recent work on *The Faculty of Speech*, p. 177, makes the following statement: "A patient with cortical motor aphasia is completely agraphic, never learns to write with his left hand, no matter how long he lives after the aphasia develops." This statement seems too sweeping, as the first part of the statement is disproven by the autopsies of the cases of Charcot, Banti, and Bramwell, and the latter part, particularly, by the case of Ogle.

These cases, it seems to me, prove beyond a reasonable doubt, that motor aphasia and agraphia are due to two distinct lesions, and may occur disassociated. They also prove that motor agraphia is not usually a symptom of a lesion limited exactly to the cortical area of Broca.

The cases reported by Broca, Tamburini,[21] and Marchi,[22] together with the notes of an unpublished case, with autopsy, sent to me through the courtesy of Dr. John K. Mitchell, of Philadelphia, furnish very strong evidence in support of my second proposition; that is, when motor aphasia and agraphia coexist, the lesion probably involves the bases of the second and third left frontal convolutions. In each case above referred to, the symptom-complex—motor aphasia, with agraphia unaccompanied with hemianopsia, alexia, or word deafness—was present. The cases of Broca and Mitchell are of such great importance in this connection that I will give a short *résumé* of each:

Broca's case, a man aged eighty-four years, came under his care October 27, 1861, with the following history: In April, 1860, he had a slight apoplectic attack, from which he recovered in a few days. No paralysis of the limbs occurred, but his speech was lost, save for four or five very simple words. His intelligence appeared normal. He understood all that was said to him and he made use of expressive gestures. He had been in this condition eighteen months prior to Broca's observation of him. To his questions he was able to reply only by signs accompanied by one or two syllables pronounced hastily and with much effort. Broca asked him to write and, although his arm and hand were not paralyzed, he could not write a single letter. His sight and hearing were good. The tongue was protruded straight and was movable in all directions. The patient died November 8, 1861. At the autopsy the lesion, an old hemorrhage, was exactly limited to the posterior third of the second and third of the left frontal convolutions. This hemorrhage was, doubtless, the cause of the apoplectic seizure which occurred eighteen months before, as the brain showed no other lesion.

Dr. Mitchell's case was a man, aged forty-four years, who was highly intelligent and well educated. His brother died of sarcoma of the brain. He had headaches for ten or twelve years, which were attributed to eye-strain. These headaches have greatly increased for the past two or three years. In August, 1899, he had an attack of difficulty in articulation, with numbness of both hands, but no loss of power. The attack lasted ten minutes. Three weeks later there occurred incoherent speech and violent twitchings, first of the right and then of the left side of the face. Later on, similar attacks appeared, which in one or two instances were followed by a semicomatose state lasting twenty-four hours. The headaches increased to an unbearable degree. In February, 1900, optic neuritis appeared. Up to this time a tumor seemed doubtful. In several attacks he had temporary loss of speech, followed by impairment of articulation, but with the consciousness of his loss of power of formulated speech slight enfeeblement of the right arm was noted. His difficulty in writing was precisely similar in character to his difficulty in speaking.

At the operation a large endothelioma, measuring 7 cm. by 8 cm., was removed. He died two hours after. The upper third of the ascending frontal and the highest part of the superior frontal gyri were not compressed by it. The lower posterior portion of the superior frontal and the whole of the posterior portions of the middle and inferior frontal convolutions, the lower two-thirds of the ascending frontal, except the extreme lowest part, were all compressed beneath it. The tumor did not extend beyond the fissure of Rolando and did not invade the brain tissue, but started apparently from the dura, pressed upon and caused thinning of the cortex and localized atrophy of the cerebrum. Autopsy and notes were made by Dr. W. G. Spiller.

The four cases above mentioned are the only ones in literature with which I am acquainted that present, clinically, motor agraphia and motor aphasia with the pathological lesion at the base of the second and third left frontal convolutions. In Broca's case there were no other symptoms present, save the inability to write and to speak, and the lesion was limited exactly to the bases of the left median and inferior frontal convolution. In Dr. Mitchell's case, the lesion, a tumor springing from the dura, involved these same-named gyri by the constant pressure causing a thinning and atrophy of the cortex without involving the white matter. The symptoms present, apart from a very slight weakness of the right arm, were motor aphasia with agraphia, and the inability to write, corresponding exactly to the inability to speak. It would be impossible to explain away these cases on the supposition of a subcortical aphasia, the lesion being so placed as to interrupt the association tract connecting the visual word area with the motor speech, or, more properly, motor writing centre, thus placing the agraphia in the category of a sensory type.

In none of the cases was hemianopsia, word deafness, mind blindness, alexia, motor or sensory paralysis present. These cases, therefore, support the theory of the separate existence of a writing centre located at the base of the second left frontal convolution.

Drs. Fränkel and Onuf,[24] from a study of 104 cases of motor aphasia with autopsies, conclude that the function of writing is independent of Broca's area, and the presence or absence of the act of writing is of no value in differentiating a subcortical from a cortical motor aphasia.

To verify my third proposition, that isolated motor agraphia, though exceedingly rare, does occur, and depends upon a lesion at the base of the second left frontal convolution in the right-handed, I direct your attention to the cases of Pitres,[24] Charcot and Dutil,[25] and Eskridge,[26] together with my own case of a glioma at the base of a second left frontal convolution, the only localizing symptom present being complete motor agraphia uncombined with any form of aphasia, in a right-handed person, without paralysis or hemianopsia.

In Pitre's case the intelligence was normal; there was paralysis of the right side without anæsthesia, which soon disappeared; there was no trace of word deafness or word blindness; nevertheless, the patient was incapable of writing spontaneously or from dictation, but could copy in fac-simile. This condition existed ten years. He had hemianopsia and was able to write with the left hand. Those who would give no credence to this case because of the presence of hemianopsia explain the loss of writing on the supposition of a subcortical lesion interrupting the association tract between the left angular gyrus and the motor area of the left side. While any explanation of the case without post-mortem evidence is purely speculative, the clinical symptoms certainly place it in the category of a partial motor agraphia. A subcortical lesion so placed as to produce hemianopsia and sensory agraphia must, necessarily, have destroyed the optic radiations as well as the fibres connecting the angular gyrus with the motor zone, and because of its size produced word blindness or alexia. The clinical record shows that word blindness did not exist and that the patient was able to copy in fac-simile. I have been able to find but six cases, with autopsies of sensory agraphia, all of which were accompanied with blindness or alexia.

The case of Charcot and Dutil was that of a woman who, after her first apoplectic attack, had no symptom except motor agraphia. Years later, a second attack left motor aphasia. After repeated attacks there developed pseudobulbar paralysis. There was no word blindness or deafness. At the autopsy a focus of disease was found in the foot of the second left frontal convolution, another at the foot of the third left frontal convolution, and three in the middle of the right hemisphere. This case seems perfectly simple if we could but prove that the oldest focus of softening was at the base of the second left frontal convolution, as the clinical symptom—motor agraphia existing alone so long a time—would indicate. The presence of agraphia for years before motor aphasia occurred certainly stamps the case as one of pure motor agraphia.

The case described by Eskridge had no paralysis of motion or sensation; no sensory aphasia. His power of articulation was perfect, except a little slow. He could talk, read printing or writing aloud or to himself, and understood what he read. There was no difficulty in propositionizing, and the movements of the lips and tongue were preserved except in uttering very long and hard words. Otherwise it was impossible to discover any speech defect. In writing he formed letters properly, but transposed letters, words, phrases, and sentences, so that it was quite impossible to read what he had written. Formerly he had been an expert penman. He was observed for two months and he tried to write a letter daily, consisting of a few lines. He spent two hours or more in writing or erasing words, before he could finish the assigned task. At times he seemed to recognize his mistakes in spelling, and at other times he did not recognize them and was indifferent to them. In trying to correct his errors he would usually make a greater one. At the operation a cyst was found at the base of the second left frontal convolution, which had destroyed the cortex over an area of about half an inch in diameter and had extended into the white substance. After the operation his writing improved very rapidly.

According to Dejerine and Collins,[27] the ideal case to prove the existence of a centre whose function is to store graphic images, and whose destruction would cause agraphia, would be one in which there was inability to write spontaneously and from dictation, and in which no disturbance of motility, particularly no disturbance of the right upper extremity, no aphasia, no alexia, no verbal deafness, and no verbal blindness existed. Collins states "that if anything is needed to make the data of aphasia more reliable in the future and more utilizable as scientific evidence in support of or in behalf of certain theories of the genesis of speech and localization, it is that the patient be studied methodically and carefully, and, when the case comes to autopsy, that the findings be recorded in scientific language, particularly the seat, extent, and nature of the lesions. A study of subcortical lesions to be of much value must be done with the microscope."

Both conditions, I submit, are fulfilled by my case, the notes of which are as follows:

The patient, Mrs. L. W., an intelligent Irish woman, received her education in the public schools. Her age was thirty-seven years, and she had been married for fifteen years; had had five living children and one miscarriage at the third month. Her father died at the age of sixty years, of cancer of the face. Mother alive and in perfect health. Has a brother and a sister living and in good health. Has lost two sisters, one of Bright's disease, the other from consumption. The patient had always enjoyed excellent health up to the onset of the present illness, which began about Christmas, 1897, when she began to fail in strength and had several attacks of vertigo, without falling and unattended with nausea or vomiting. A short time afterward she developed severe frontal and occipital headaches, with an occasional attack of vomiting. The headaches continued with increasing severity until the fatal issue. In March, 1898, she noticed that her sight was failing and that she could not read print without the use of glasses. At about the same time she began to complain of a numbness in the right arm. At no time during her illness did she have a unilateral or general convulsion. Her bowels have been rather constipated. She has had perfect control over her bladder. Has menstruated regularly and in a normal manner. Patient converses intelligently, makes no errors, and her memory for past and recent events is good. At no time during illness has she had the slightest difficulty in giving expression to her thoughts by articulate speech.

Examination, April 5, 1898. Patient 5 feet 4 inches in height; weight, 140 pounds. Physical examination of the chest and abdomen elicited nothing abnormal. Urine normal. Temperature 98.6° F., pulse 68. Head well formed and perfectly symmetrical. No evidence of previous injury; no exostosis. Percussion of scalp elicits no tenderness. Veins of forehead not prominent. Pupils moderate and equally dilated, and respond actively to light and accommodation. Wernicke's hemianopic pupillary phenomenon not present. No hemianopsia. Optic neuritis of both eyes, though more intense in right eye. Owing to paresis of the right external rectus muscle, the right eyeball could not be rotated outward, but its movements upward, downward, and inward were found normal. Diplopia existed. Excursion of left eyeball normal. Color vision normal. Drum membranes appeared normal. Hearing distance for watch in each ear was three feet. Movements of tongue normal. No fibrillation or atrophy. Sense of taste and smell normal. Soft palate and uvula move in a normal manner. No loss of sensation over buccal or pharyngeal mucous membranes. Movements of vocal cords normal. No difficulty in swallowing. No stiffness of neck muscles or those of the spine. Both sides of the face symmetrical. No evidence of facial paralysis. Patient is able to perform, in a normal manner, symmetrical bilateral movements. Grip of right hand is slightly weaker than that of left. Dynamometer: outer scale, right, 50; left, 75. No muscular atrophy anywhere discoverable. No stiffness of muscles. Fine and coarse movements of both hands and arms normal. Movements of lower extremities normal. Muscular power of both legs equal. The most careful examination of the skin of the entire body showed no sensory disturbances. No œdema or other vascular changes. The most careful reinforcement failed to elicit the presence of patellar tendon reflexes. No ataxia in upper or lower extremities. The sense of posture normal. Romberg's symptom absent. Gait perfectly normal. She has always been right-handed.

Speech. Patient recognizes and names correctly any object placed before her. She can recognize and accurately name, with her eyes closed, any familiar object when placed in her right hand, such as coins and the like. She understands perfectly what is said to her. She recognizes familiar hymns. She can repeat words after another and speak voluntarily without the slightest hesitation, and always correctly. She can read correctly, with the aid of glasses, ordinary sized print or writing. She recognizes numerals at once. The only difficulty present is the total inability to write. Although she understands perfectly written language and can read to herself or aloud, she cannot write, voluntarily, nor form, correctly, a single letter, and cannot write from dictation or copy. She holds the pen in a perfect manner, and performs movements with it as if to write. Her writings consisted, however, of nothing more than a series of united curves. She was unable to write with her left hand, as she was not ambidextrous. The fine and coarse muscular movements of the right hand are executed in a perfect manner, and there exists no paralysis of the muscles of the hand, forearm, or arm. Before the present illness began, she could write perfectly. She showed me some of her writing, which was excellent.

Without the slightest external manifestation of a syphilitic infection, it was thought wise to give the patient the benefit of the doubt, and hence potassium iodide combined with mercury was administered for six weeks.

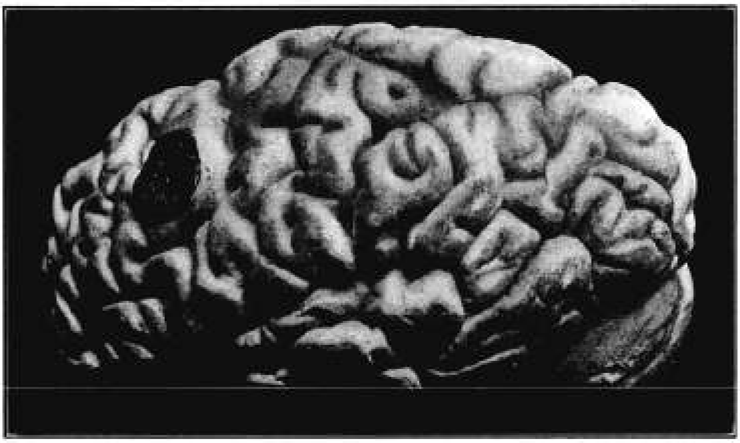
May 20*th*. After six weeks' continuous use of increasing doses of potassium iodide combined with mercury it was discontinued, there having been no amelioration of any symptom. No sensory or motor symptoms. No hemianopsia or alexia. Voluntary speech perfect. Cerebration, which was very active at my first examination (the patient answering the numerous questions put to her very quickly and correctly), was now quite slow. This slowness of speech was not in any sense aphasic in character, as all questions were answered correctly, only there was considerable deliberation before the answers came. Memory good. There is an absolute inability to write voluntarily from dictation or to copy. The patient holds the pen or pencil in perfect position and the movements of the forearms and hands are normal. Has perfect control of sphincters. In no other particular was her condition different from that noted above, save that there is a tendency to fall or to stagger toward the right side. This ataxia did not exist at the date of the first examination. The patient has noticed this condition for about three weeks, and she thinks it is becoming gradually worse. It is an inco-ordination similar to that of cerebellar disease, and is like that frequently observed in disease involving the prefrontal lobe.

This condition of complete agraphia continued throughout her illness and was, with the exception of a gradual development of a slow cerebration and frontal ataxia (Bruns), the only localizing symptom present. At no subsequent time was there found the slightest evidence of motor or any form of sensory aphasia. She never had a partial or complete unilateral or general convulsion, never became paralyzed or developed any sensory disturbance. Hemianopsia or alexia were never present. The presence of agraphia without motor aphasia, with a gradually increasing slow cerebration and ataxia, coupled with double optic neuritis, with severe and continuous headaches, unaccompanied with alexia, led to the diagnosis of a lesion of the prefrontal lobe, probably a new-growth, the situation of which I was led to believe, owing to the fact that the slow cerebration and inco-ordination developed subsequently to the agraphia, was at the base of the second frontal convolution of the left side.

After six weeks of further suffering, there being no change in her symptoms, she consented to an exploratory operation, which was performed July 19, 1898, by Dr. E. D. Ferguson, of Troy, N. Y. The left Rolandic area was exposed after the method of Reid, under strict asepsis. The trephine opening was next made through the middle third of this area. On removing the button of bone the dura bulged into and through the opening, forming a very elastic swelling which pulsated feebly. On incising the dura there was an escape of a considerable amount of subarachnoid fluid, followed immediately by the formation of a troublesome hernia cerebri. The opening was now enlarged forward by the rongeur forceps, and a careful search was made for a lesion, but none was detected. The cortical area, exposed by trephine opening, appeared normal. While discussing the propriety of enlarging the opening still more forward, her condition became so alarming that it was decided to close the opening at once. The vessels having been secured, the wound was closed with kangaroo tendon and a gauze drain was left in the most dependent part.

July 20, 1898. On awakening from her etherization, it was discovered that her right arm was paralyzed. She appeared very drowsy and stupid, and complained of great pain in the region of the wound. She was unable to move the right arm, and the movements of the right leg were impaired.

21st. Patient rapidly passed into a condition of unconsciousness. No control over bladder. Pulse rapid and very feeble. Temperature 101° F. She died at 4 P.M.



Left cerebral hemisphere, showing exact location and size of tumor producing motor agraphia in Dr. Gordinier's case.

Autopsy, July 22, 1898, at 10.30 A.M. Only the head permitted to be examined. Trephine opening, 4.5 cm. by 4.75 cm. Elevation of hernia cerebri above dura, 2 cm. Dura everywhere free. Superior longitudinal sinus contained a soft, recent thrombus. Vessels of pia injected. Left cerebral hemisphere: Marked hemorrhagic softening of the hernia cerebri and surrounding brain tissue. Pia not adherent save at location of the new-growth, which was found occupying the foot of the second left frontal convolution, being distinctly separated from the arm area by the precentral sulcus, which sulcus in this case was unusually well defined. The growth was elevated about 0.5 cm. above the surrounding cortex, was slightly irregular in outline, of a distinctly firm consistency, and oval in shape. Its longest cortical diameter was 2 cm. The pia was intimately adherent to it. On section, the tumor was seen to extend downward and inward in the centrum semiovale as far as the roof of the anterior cornu of the lateral ventricle, and forward to near the apex of the frontal lobe, involving almost exclusively the white matter of the second frontal gyrus. The left third or inferior frontal convolution through its whole extent was found (macroscopically) perfectly normal, as was its associated centrum ovale. The centrum ovale of the superior first frontal convolution was, toward its ventral part, infiltrated by the growth. The motor convolutions in their middle third formed a part of the hernia cerebri. This area was filled with minute capillary hemorrhages, the result of the sudden relief of the intercranial pressure. The rest of the motor area and the underlying white matter was found normal. The supramarginal and angular gyri, with their associated white matter, was very carefully examined and found normal, as were the convolutions and white matter of the occipital and temporal lobes. The basal ganglia and the internal capsule showed no naked-eye changes. The convolutions, centrum ovale, basal ganglia, and internal capsule of the right cerebral hemisphere normal. The interior of the skull was found normal.

Histological Examination. The histological examination of the tumor showed it to be a glioma of rapid growth. It was of a yellowish-gray color, rather firm in consistency, not very vascular, and was inseparably blended with the surrounding white matter. It presented no softened areas, and there was no softening of the white matter in advance of it. It was not at all cystic in character. Sections of the tumor were placed for four days in Orth's fluid and subsequently hardened in alcohols of increasing strength. Small cubes were embedded in celloidin and stained with hæmatoxylin and eosin, Von Gieson's picric acid, fuchsin and hæmatoxylin, Weigert's myelin stain, and with Mallory's phosphomolybdic acid, hæmatoxylin, and phosphotungstic acid hæmatoxylin.

Microscopic examination of the left inferior or third frontal convolution, with the associated white matter, showed it to be normal.

The ventricles contained an excess of fluid. The brain stem, pons, cerebellum, medulla, and the cranial nerves showed no macroscopic changes.

This case seems to prove the existence of a separate and distinct cortical centre for writing, having the same relation to writing movements as the motor speech centre has to speech movements. This centre is located at the base of the second left frontal convolution for the right-handed, and probably in the same location in the right hemisphere for the left-handed. Destruction of this centre produces pure motor agraphia without aphasia or paralysis of either arm.

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A CONTRIBUTION TO THE SURGERY OF THE BRAIN, WITH THE REPORT OF TWO CASES OF TUMORS OF THE CEREBELLUM WHICH WERE LOCATED AND ON WHICH OPERATIONS WERE PERFORMED.

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Case I.—*Cyst of cerebellum located in the right lobe, in which two operations were performed, with ultimate recovery.*

History. Julia R., aged nine and one-half years. Family history good. Father healthy, strong, and well nourished. Mother very healthy and strong. The patient was one of twins, the other dying at birth. Both had supernumerary little fingers. The brothers and sisters are all in perfect health, the youngest children being twins, also with supernumerary little fingers. Father by a previous marriage had several children, two of the daughters dying of rapid pulmonary tuberculosis during the winter of 1894–95. The other children by the former marriage are healthy and apparently not tuberculous. The two daughters who died of phthisis lived in the same house with the family.

The patient, up to the summer of 1895, was very healthy and active. She was well developed and strong, and the brightest of the children. During the summer she began to complain of headache, which she said was in her eyes and forehead; it was occasionally so severe as to cause her to cry. Toward December these attacks became very frequent, and compelled her to remain in the house most of the time. The location of the pain was changed, and was now in the back of the head, and down the back of the neck, on the right side. During this period she vomited occasionally, always just as she got out of bed in the morning. She did not complain of nausea, but had frequent attacks of vertigo, which she called "swimming in the head." These attacks were usually in the morning when she was getting up. Her parents do not remember having noticed any clumsiness or staggering during this time; but from being the errand girl of the household, she became inactive and disposed to lie in bed. When not suffering, her parents thought her mind was as active and bright as it had ever been.